APPENDIX 11

STATEMENT OF
RICHARD J. FALK, M.D.

1. I have been engaged in the field of medicine, specifically obstetrics, gynecology and reproductive endocrinology and pathology for the past forty years. I have been engaged in the evaluation, investigation and research of hormones and pharmaceuticals. I have reviewed much of the world's literature as to the safety and efficacy of DES (Diethylstilbestrol) when used in pregnancy. I have been engaged in research in the field of hormones, specifically reproductive hormones, engaged in conferences regarding reproductive medicine, I conducted clinical trials for pharmaceutical manufacturers, and served as an advisor to the Food and Drug Administration in reproductive endocrinology. I have served as a consultant to the National Institute of Health and held academic positions in the field of obstetrics, gynecology and reproductive endocrinology. My Curriculum Vitae is attached hereto.

2. If called to the trial of this case, I will testify that:

a. In 1953, a study (Dieckmann) was published, which was the culmination of a long line of prior literature questioning the efficacy of DES and pregnancy. Subsequent to this study, there was no rational support for the use of DES in pregnancy. In addition, the medical literature raised significant questions as to the risks dangers to the future pregnancy function of the developing female, exposed in utero to DES. This literature could have and should have alerted a reasonably prudent pharmaceutical seller to the need for further human and animal testing, and investigation of DES in pregnancy, and the danger of promoting the drug for use to pregnant women without undertaking further generational testing. Had appropriate animal and human testing and investigation of this issue been conducted the dangers of prenatal exposure to DES daughters and their pregnancy function impairment would have become apparent and the drug would have and should have been recalled for use in pregnancy.

Thereafter, any manufacturer who promoted the use of DES to prevent the accidents of pregnancy failed to adequately warn the medical profession and the Food and Drug

Administration that the risks of DES to the exposed female outweighed the benefit and that such information was a misrepresentation of the state-of-the-art and that the product, without proper labeling, was dangerous and defective.

3. The defendant's failure to conduct these tests was below the accepted standards of prudent and careful pharmaceutical practice. Had the drug companies conducted prerequisite investigation, they would have discovered that DES was a teratogen and that it affected the reproductive potential of the exposed daughter and posed a foreseeable risk of a miscarriage, infertility, ectopic pregnancy and preterm delivery.

4. It is my opinion, based upon my education, training and research experience, that DES exposure in utero is an obstacle to normal fertility, increases abnormal progesterone receptors, and causes small and dysplastic uteri in the exposed daughter.

5. The basis for my opinion as to the practices, procedures, responsibilities, and obligations of pharmaceutical manufacturers comes from my experience as a physician and consultant to the National Institute of Health, pharmaceutical manufacturers, as well as my education, training, and experience as an obstetrician, gynecologist and reproductive endocrinologist, as well as symposia, teaching, and professional activities.

6. The following principles were well known in the scientific community before 1953:

a. Animal models in proper strains existed which showed transplacental DES risks to the exposed female fetus reproductive tract suggestive of pregnancy dysfunction;

b. Hormones, estrogen, DES, and Other agents were reported to have transplacental transport ability in animals and humans creating offspring toxicity;

c. Synthetic estrogen (DES) was reported to cause malformation, metaplasia and dysplasia in relevant animal species.

d. Some drug companies were conducting offspring studies on drugs prior to marketing;

e. Synthetic estrogen (DES) was known to affect specific reproductive target organs (i.e. uterus and cervix) in the daughter of the recipient;

f. Prenatal exposure to estrogen caused intersexuality in relevant test animals - - feminization of males, masculinization of females, and other malformations of the reproductive tract (DES is an estrogen); and

g. Synthetic estrogen (DES) in the dosage regimens recommended to humans during pregnancy were hundreds of times the previous recommended therapeutic levels recommended for use to affect reproductive organs in the recipient.

7. I have reviewed the relevant sections of the defendant's literature which omits any meaningful reference to the 1939 Raynaud study (intersexuality in mice), and the 1939 Greene, Burrill and Ivy Study (feminization, gonadal, vaginal and sexual tract retardation and changes in offspring exposed in utero to DES), the 1940 Zuckerman synopsis of estrogen's teratological effects on the reproductive tract, or the 1949 Burrows study (newborn females exposed in utero to estrin born with cornification of the vagina and distended uterine horns). These omissions were substantial and the inclusion of these risks would have warned the government and the medical profession of these risks and most likely would have decreased or prevented the use of this drug by pregnant women. As time elapsed after 1953, more and more reports of the then risks and dangers of synthetic estrogens appeared culminating in the 1962 thalidomide disaster, after which it was gross and wanton negligence to recommend DES to pregnant women.

8. The Eli Lilly and Company, as a major manufacturer, promoter and distributor of DES, compared to the other drug companies, failed to conduct adequate follow-up studies, in animals or humans, to determine the long-term delayed effects of DES on the sexual tract of the

exposed fetus. These failures of Lilly and others to conduct adequate long-term generational animal testing designed to investigate or reproduce the transplacental effects from DES was a departure from prudent standards of pharmaceutical, industry standards as they existed at that time in America.

9. The drug houses selling DES should have tested it to study, determine, evaluate, demonstrate, or reproduce toxicity of the teratological nature, whether immediate or delayed, to the female offspring, whether human or animal, exposed to DES in pregnancy. Adequate investigation of the questions raised by the literature would have revealed the risks to the pregnant daughter previously exposed in utero and to her subsequent offspring due to premature delivery.

10. With evidence linking the action of DES to reproductive tract changes in the offspring of DES in exposed animals, and in the presence of literature relating reproductive organ anomalies in offspring exposed to DES, a reasonably prudent manufacturer should have undertaken studies indicated by the Van Winkle rules (JAMA, 1944) designed to fully evaluate the potential toxicity of DES, including:

a. Complete literature reviews of all data relating to DES and the development of reproductive organ anomalies in offspring exposed to DES:

b. Complete literature review of other hormone related reproductive organ anomalies in offspring exposed to those agents; and

c. Complete and thorough clinical, laboratory, tissue and autopsy review of each case of offspring reproductive organ anomalies associated with DES and other hormone exposure in utero.

11. DES as labeled and with its then warnings, was an unreasonably, dangerous and defective product in the mid-1950's.

I state under penalty of perjury that the above statement is true and correct. Executed this 27 day of August, 2004.

DATED: 8/27/2004

Richard J. Falk, M.D.